

Magnesium, zinc, and chromium nutriture and physical activity¹⁻³

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ABSTRACT Magnesium, zinc, and chromium are mineral elements required in modest amounts to maintain health and optimal physiologic function. For physically active persons, adequate amounts of these micronutrients are needed in the diet to ensure the capacity for increased energy expenditure and work performance. Most physically active individuals consume diets that provide amounts of magnesium and zinc sufficient to meet population standards. Women tend to consume less of these minerals than is recommended, in part because they eat less food than men. Inadequate intakes of magnesium and zinc have been reported for participants in activities requiring restriction of body weight. Dietary chromium is difficult to estimate because of a lack of appropriate reference databases. Acute, intense activity results in short-term increases in both urine and sweat losses of minerals that apparently diminish during recovery in the days after exercise. Supplemental magnesium and zinc apparently improve strength and muscle metabolism. However, evidence is lacking as to whether these observations relate to impaired nutritional status or a pharmacologic effect. Chromium supplementation of young men and women does not promote muscle accretion, fat loss, or gains in strength. Physically active individuals with concerns about meeting guidelines for nutrient intake should be counseled to select and consume foods with high nutrient densities rather than to rely on nutritional supplements. The indiscriminate use of mineral supplements can adversely affect physiologic function and impair health. *Am J Clin Nutr* 2000;72(suppl):585S-93S.

KEY WORDS Trace elements, endurance, muscle strength, physical activity, nutritional status, mineral supplements, magnesium, zinc, chromium

INTRODUCTION

Persons who seek to promote their health generally rely on 2 behaviors, consuming a proper diet and engaging in more physical activity (1). Increasingly, there is an emphasis on the synergistic relation between diet and exercise for well-being and a growing awareness of the beneficial role that mineral element nutrition may play in achieving good health and enhanced physiologic function.

In contrast with macronutrients, which are consumed in large amounts (hundreds of grams daily), micronutrients, such as magnesium, zinc, and chromium, are ingested in very small amounts (micrograms and milligrams per day). The macronutrients provide sources of energy (carbohydrate and fat) required

to fuel the body during work, to maintain hydration (water), and to provide the body structure (protein) for performing work. Magnesium, zinc, and chromium, despite their relative paucity in the diet and the body, perform important roles in regulating whole-body metabolism, including energy utilization and work performance.

The importance of these micronutrients is revealed by the diversity of metabolic processes they help to regulate. Magnesium, a ubiquitous element that plays a fundamental role in many cellular reactions, is involved in >300 enzymatic reactions in which food is catabolized and new chemical products are formed (2). Examples include glycogen breakdown, fat oxidation, protein synthesis, ATP synthesis, and the second messenger system. Magnesium also serves as a physiologic regulator of membrane stability and is involved in neuromuscular, cardiovascular, immune, and hormonal function (3-5).

Zinc, also an intracellular cation, is required to serve either a catalytic or structural role by >200 enzymes in mammals. Zinc-containing enzymes participate in many components of macronutrient metabolism and cell replication. In addition, some zinc-containing enzymes, such as carbonic anhydrase and lactate dehydrogenase, are involved in intermediary metabolism during exercise. Another zinc-containing enzyme, superoxide dismutase, protects against free radical damage (6).

The element chromium is the subject of growing interest in the public and scientific communities. Mammals need trivalent chromium to maintain balanced glucose metabolism (7), and thus chromium may facilitate insulin action (8, 9). This insulinogenic characteristic of chromium has prompted the hypothesis by Evans (10) that chromium has an anabolic function.

Persons who seek to optimize the benefit of nutrition and physical activity on health and function may resort to using mineral supplements. Proponents of supplementation cite the burgeoning evidence that magnesium, zinc, and chromium play vital roles in facilitating the transduction of chemical energy in food

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TABLE 1
Estimates of the percentage of individuals who do not consume 100% of the dietary reference intake of magnesium (12) and the recommended daily allowance for zinc (13)¹

Sex and age (y)	Magnesium	Zinc
	%	
Men		
20–29	61	62
30–39	62	58
40–49	57	63
50–59	66	70
60–69	65	72
>70	76	86
>20	63	66
Women		
20–29	86	80
30–39	76	80
40–49	73	80
50–59	77	85
60–69	80	90
>70	79	88
>20	78	83

¹ Adapted from the US Department of Agriculture Continuing Survey of Food Intakes by Individuals (11).

into potential energy for work and for integrating physiologic functions to enhance physical performance. They also refer to the results of nationwide surveys of food intake that suggest that most Americans consume inadequate amounts of essential minerals (11). On average, >60% of US men and women aged ≥20 y consume less than the dietary reference intake (DRI) for magnesium (12) and less than the recommended dietary allowance (RDA; 13) for zinc (**Table 1**). Similarly, the usual intake of chromium from self-selected diets is only 33 and 25 μg/d for men and women, respectively (14), compared with the recommended safe and adequate daily intake of 50–200 μg (13).

This paper addresses the need for magnesium, zinc, and chromium supplements in physically active persons by examining the following questions: Are dietary intakes of these minerals adequate in physically active persons? What is the evidence that increased physical activity negatively affects mineral nutritional status? Is there a measurable benefit of physiologic amounts of magnesium, zinc, or chromium supplementation on work performance? What are the potentially adverse effects of generalized mineral supplementation on health and function?

MAGNESIUM

Interest in potential adverse effects of physical activity on magnesium status among sports medicine specialists began with the diagnosis of magnesium deficiency in a female tennis player who had frequent episodes of muscle spasms associated with prolonged outdoor exercise (15). In the presence of otherwise normal physical, neurologic, and blood biochemical findings she had decreased serum magnesium (0.65 mmol/L; normal range: 0.8–1.2 mmol/L). The muscle spasms were resolved after a few days of oral magnesium treatment (500 mg/d).

Dietary intake

Estimates of magnesium intakes of individuals involved in a variety of types of physical activity are available (**Table 2**). Mag-

nesium intakes among male and female collegiate athletes were found to equal or exceed 66% of the DRI. Men participating in collegiate football had greater dietary magnesium intakes than did female collegiate gymnasts and basketball players (16, 17). Male soccer players who used institutional food services generally exceeded the DRI for magnesium intake (18). Recreational triathletes also reported adequate dietary magnesium (19). On average, most athletes had magnesium intakes that approximated 70% of the DRI. Although these initial findings suggest that dietary magnesium may be adequate by population standards for these groups, they do not answer whether physically active individuals have magnesium intakes that differ from those of their less-active counterparts.

Limited data (20) describe magnesium intakes among physically active and control subjects (**Table 3**). In a small sample of female collegiate runners, eumenorrheic runners had greater magnesium intakes than did amenorrheic control subjects (21). A positive trend existed between energy and magnesium intakes in these women. These limited findings suggest that dietary magnesium may be limiting among some physically active women.

In a large survey, Fogelholm et al (22) found greater magnesium intakes among 114 male Finnish athletes than 117 age-matched, male control subjects. Similarly, dietary magnesium was greater among Nordic skiers than in their age- and sex-matched, nontraining counterparts (23). This difference may be attributed to the skiers' greater energy intake (men: 1.6 compared with 1.2 MJ/d; women: 1.2 compared with 0.9 MJ/d) and the greater nutrient density of the skiers' diet (423 compared with 339 mg/MJ and 398 compared with 378 mg/MJ for men and women, respectively). Regardless of activity status, dietary magnesium exceeded the recommended intakes in both studies. Thus, physical activity per se apparently does not predispose individuals to inadequate magnesium intake.

Longitudinal monitoring of diet during physical training has also shown that physically active persons consume adequate dietary magnesium (24). Successful collegiate female swimmers had dietary magnesium intakes similar to those of nontraining women both before (283 compared with 263 mg/d) and at the end (329 compared with 251 mg/d) of the competitive season; these

TABLE 2
Dietary magnesium intake among selected groups of physically active adults¹

Study and physical activity	Dietary magnesium
	mg/d (% of DRI)
Hickson et al, 1986 (16)	
Basketball (<i>n</i> = 13 F)	204 (66)
Gymnastics (<i>n</i> = 9 F)	204 (66)
Hickson et al, 1987 (17)	
Football (<i>n</i> = 11 M)	277 (69)
Hickson et al, 1986 (18)	
Soccer (<i>n</i> = 18 M) ²	469 (117)
Soccer (<i>n</i> = 18 M) ³	361 (90)
Worme et al, 1990 (19)	
Triathlon (<i>n</i> = 21 F)	324 (105)
Triathlon (<i>n</i> = 50 M)	362 (91)

¹ DRI, dietary reference intake: 310 mg/d for women and 400 mg/d for men aged 19–30 y (12).

² Preseason.

³ Postseason.

TABLE 3Reported energy and magnesium intakes among physically active and control individuals¹

Study and physical activity	Dietary magnesium mg/d (% of DRI)
Zierath et al, 1986 (21)	
Running (<i>n</i> = 8 F) ²	164 (53)
Running (<i>n</i> = 9 F) ³	276 (89)
Control (<i>n</i> = 7 F)	92 (30)
Fogelholm et al, 1991 (22)	
Athletics (<i>n</i> = 114 M)	548 (137)
Control (<i>n</i> = 117 M)	436 (109)
Fogelholm et al, 1992 (23)	
Skiing (<i>n</i> = 5 M)	646 (162)
Control (<i>n</i> = 19 M)	407 (99)
Skiing (<i>n</i> = 7 F)	478 (155)
Control (<i>n</i> = 20 F)	304 (96)

¹DRI, dietary reference intake: 310 mg/d for women and 400 mg/d for men aged 19–30 y (12). Adapted from Lukaski (2).²Amenorrheic runners.³Eumenorrheic runners.

intakes exceeded the DRI for the swimmers (92% and 106%) and were similar to the DRI for the control subjects (85% and 81%).

Physical activity and biochemical evidence of magnesium deficiency

Serum magnesium concentration, although commonly used to measure magnesium nutriture in nutritional surveys of physically active persons, is a relatively insensitive index of marginal magnesium status (3). Indeed, its insensitivity generally rules out a conclusion that physical activity does not adversely affect magnesium status. On the other hand, we know from the work of Fogelholm et al (22) that serum magnesium is in the normal range when intake is adequate, irrespective of physical activity.

In contrast with the relatively static serum concentrations of magnesium, researchers have observed substantial redistributions within the body and increased losses of magnesium from the body in response to acute bouts of exercise. Compared with preexercise conditions, magnesium shifts from the plasma into the red blood cells (25). In a study by Deuster et al (25), the direction and magnitude of magnesium redistribution in the circulation was influenced by the intensity of the preceding exercise; the greater the energy requirement from anaerobic or glycolytic metabolism, the greater the translocation of magnesium from the plasma into the red blood cells. In addition, urinary excretion of magnesium increased 21% (131.5 compared with 108.6 mg/d; $P < 0.05$) on the day of the exercise compared with the preceding nonexercise day. Compensation for these alterations in magnesium took place the day after exercise when plasma, red blood cell, and urinary magnesium concentrations returned to preexercise values (25, 26). Thus, homeostatic mechanisms accommodate the transient loss of magnesium with a return to equilibrium during recovery from the previous exercise bout. In addition, Deuster et al (25) found general relations between the change in urinary magnesium excretion and oxygen uptake normalized for body weight ($r = 0.84$, $P < 0.001$) and postexercise blood lactate concentration ($r = 0.68$, $P < 0.01$).

Another route of magnesium loss during exercise is sweat and cellular exfoliation. Men performing controlled work for 8 h on

ergocycles in the heat (100°F) lost 15.2–17.8 mg Mg/d in sweat (27). In this study, magnesium losses in sweat accounted for 4–5% of daily magnesium intake and 10–15% of total magnesium excretion (feces, urine, and sweat).

Magnesium supplementation and physical performance

Use of magnesium supplements has been reported to affect cellular metabolism. Golf et al (28) gave female athletes with plasma magnesium concentrations at the low end of the range of normal values either a magnesium supplement (360 mg as magnesium aspartate) or a placebo each day for 3 wk. The supplemented athletes had lower activities of serum total creatine kinase and creatine kinase isoenzyme from skeletal muscle after training than did the women given the placebo. Also in this study, compared with placebo-treated rowers, competitive rowers consuming a magnesium supplement (360 mg/d) for 4 wk had lower serum lactate concentrations and 10% lower oxygen uptake during a controlled submaximal exercise test (28). Similarly, moderately trained adults given either placebo or magnesium supplements (250 mg Mg/d as magnesium picolinate) had improved cardiorespiratory function during a 30-min submaximal exercise test (29). These findings suggest a potentially beneficial effect of magnesium supplementation on muscle metabolism and work efficiency.

Magnesium supplementation also has been shown to improve muscle function. Brilla and Haley (30) assigned young men who were matched for quadriceps strength and were participating in a 7-wk strength-training program to receive either a placebo or a magnesium supplement (magnesium oxide) to reach a total daily magnesium intake (diet plus supplement) of 8 mg/kg body weight (30). Total daily magnesium intakes were estimated to be 507 and 250 mg/d for the men receiving the magnesium supplement and placebo, respectively. Peak knee-extension torque increased more in the magnesium-supplemented than in the placebo-treated men. This finding suggests a beneficial role for magnesium in the performance of activities that require a predominant contribution of glycolytic metabolism.

Magnesium supplementation of young men involved in different types of physical training failed to promote enhanced performance (31). Active men participating in a 12-wk exercise program of predominantly aerobic or a combination of aerobic and anaerobic activities consumed either a magnesium supplement (250 mg/d) or placebo. Peak oxygen uptake increased in men in both training programs; there was no beneficial effect of magnesium supplementation on performance. Training increased plasma magnesium concentrations in the aerobic-anaerobic group but had no significant effect on plasma magnesium in the aerobic group. Increased physical activity decreased erythrocyte magnesium concentration independently of supplementation and type of activity. Urinary magnesium loss did not change with magnesium supplementation or training. These findings indicate that magnesium supplementation does not exert an independent effect on performance gains when serum magnesium is within the range of normal values and confirm that changes in plasma magnesium depend on the type of metabolic stressor imposed by the physical training (anaerobic or aerobic).

ZINC

The effects of severe zinc deficiency on skeletal muscle are well defined (32). Rats fed diets very low in zinc exhibit reduced



TABLE 4Estimated zinc intake of physically active and control adults¹

Study, physical activity, and subject sex	Dietary zinc mg/d (% of RDA)
Lukaski et al, 1983 (36)	
Football, basketball, hockey, track and field (male)	16.3 ± 1.5 ² (109)
Control (male)	13.7 ± 1.7 (91)
Deuster et al, 1989 (37)	
Running (female)	10.3 ± 0.7 (86)
Control (female)	10.0 ± 0.7 (83)
Lukaski et al, 1990 (24)	
Swimming (female)	12.4 ± 0.8 (103)
Control (female)	9.8 ± 0.9 (82)
Swimming (male)	17.9 ± 1.0 (120)
Control (male)	15.2 ± 1.0 (100)
Worme et al, 1990 (19)	
Triathlon (male)	13.7 ± 0.8 (91)
Triathlon (female)	10.6 ± 0.8 (88)
Fogelholm et al, 1992 (23)	
Skiing (female)	15.8 ³ (132)
Control (female)	10.5 (88)
Skiing (male)	21.9 (146)
Control (male)	14.1 (94)
Snook et al, 1995 (38)	
Running ⁴ (female)	8.7 ± 1.5 (73)
Running ⁵ (female)	9.7 ± 0.9 (81)
Control (female)	8.7 ± 0.7 (73)

¹RDA, recommended dietary allowance: 12 mg/d for women and 15 mg/d for men aged 19–30 y (13).

² $\bar{x} \pm \text{SE}$.

³Mean.

⁴Long distance.

⁵Sprinting.

muscle growth and decreased muscle DNA concentrations. Rehabilitation of protein-energy malnourished mice and children with supplemental zinc and energy fails to overcome deficits in muscle development. Animal experiments have shown the importance of zinc for skeletal muscle performance and resistance to fatigue (33, 34).

Information on the zinc needs of physically active individuals is limited (35). Practical limitations, including lack of sensitive indicators of zinc status and the difficulty of conducting controlled human studies to ascertain the interaction between dietary zinc and performance, contribute to the dearth of useful information. A variety of studies with different experimental designs provide some insight into the relation between dietary zinc and physical activity.

Dietary intake

Surveys of self-reported food and beverage intakes indicate that groups of physically active individuals generally consume zinc in amounts exceeding 70% of the RDAs of 15 and 12 mg/d for men and women (13), respectively (Table 4). However, a significant proportion of participants in some activities, such as long-distance running and gymnastics, may consume <10 mg Zn/d. Marginal zinc intake is more prevalent among women than men because women generally consume less food than do men. Voluntary food restriction, however, is characteristic of groups of active individuals who participate in activities (eg, wrestling, dancing, and gymnastics) in which physical appearance is a component of performance evaluation (36–40).

Physical activity and zinc status

Assessment of zinc nutritional status has relied on an indirect tool: serum or plasma zinc concentration. Although this approach has been successful for identifying individuals with severe zinc deficiency, it is a relatively insensitive and nonspecific indicator of marginal zinc nutriture; thus, moderate and mild zinc deficiency are difficult to demonstrate (35, 41).

Interest in the possible adverse effects of physical activity on zinc status began with the observation by Dressendorfer and Sockolov (42) that some endurance runners had significantly lower serum zinc concentrations than did men who were not participating in chronic exercise. More than 20% of the 76 male runners had serum zinc concentrations <11.5 $\mu\text{mol/L}$, the lower limit of the range of normal values. Among these runners, serum zinc concentrations were inversely related to the distances run in training. Dressendorfer and Sockolov speculated that inadequate dietary zinc and increased zinc losses in sweat might contribute to observed decreased serum zinc concentrations.

Similar findings of reduced circulating zinc were reported for some, but not all, groups of physically active individuals. In a survey of athletes, Haralambie (43) found no significant difference in serum zinc concentration between the athletes and sex-matched control subjects. Some of the athletes, however, had serum zinc concentrations <12 $\mu\text{mol/L}$. Among female runners, plasma zinc concentrations were concentrated at the low end of the range of normal values, with >20% of the values <12 $\mu\text{mol/L}$ (37, 44). In contrast, no significant differences in plasma zinc concentrations were found in comparisons of physically active collegians with sex- and age-matched, nontraining, control subjects (24, 36).

The basis for the apparent adverse interaction between physical activity and circulating zinc is not clear. One explanation is that depressed circulating zinc is associated with inadequate dietary zinc (Table 4). Among female runners, mean plasma zinc concentrations were within the range of normal values (37), but 29% of the women had plasma zinc concentrations <12 $\mu\text{mol/L}$ and 50% consumed <12 mg Zn/d. In our laboratory, mean plasma zinc concentrations were not significantly different between female swimmers and nontraining control subjects (24). Importantly, the self-reported zinc intake was <12 mg/d for \approx 15% of the women swimmers in each group. Thus, diet, rather than physical activity per se, apparently explains the hypozincemia seen in some physically active individuals.

Exercise is known to acutely change circulating zinc concentrations. Brief, intense, and prolonged endurance exercise increases plasma and serum zinc concentrations immediately after exercise (45, 46). The magnitude of the increase in plasma zinc concentration during exercise cannot be attributed to hemoconcentration (45), but may be explained by the movement of zinc from contracting skeletal muscle into the extracellular fluid because of muscle breakdown (47). After the exercise bout, plasma zinc concentration generally decreases within a brief period of time. McDonald and Keen (32) speculated that the rapid postexercise decrease in plasma zinc concentration is associated with an increased urinary excretion of zinc coupled with a redistribution of zinc from the plasma into the liver. The movement of zinc from the plasma into the liver is considered to be a consequence of the acute-phase response that is modulated by cytokines (48).

Physical activity that involves soft tissue trauma also affects plasma zinc concentration. Singh et al (49) observed reductions in plasma zinc in military personnel involved in a 5-d, intensive

training course, despite dietary intake of zinc during the training (49). The decreased plasma zinc concentration was associated with increased plasma IL-6 (interleukin-6) concentrations. The authors attributed the reductions in plasma zinc concentration primarily to a redistribution of zinc into the liver as a consequence of metallothionein synthesis stimulated by IL-6. Similarly, Lichton et al (50) reported that male soldiers engaged in a 34-d field exercise at 1829 m experienced a significant decrease in plasma zinc concentration. In this study, the soldiers on maneuvers had a greater zinc intake than did the sedentary soldiers who were used as control subjects; zinc intakes of both groups of soldiers exceeded the recommended intake of 15 mg/d. Urinary zinc losses by the active soldiers increased from 6.1 $\mu\text{mol/d}$ (400 $\mu\text{g/d}$) under relatively inactive conditions to $\approx 10.7 \mu\text{mol/d}$ (700 $\mu\text{g/d}$) under field conditions. Urinary zinc loss under these conditions represents $\approx 6\%$ of recommended daily zinc intake.

Increased urinary zinc excretion after acute exercise has been reported. Studies of men participating in brief or prolonged periods of exercise found a 50–60% increase in urinary zinc loss over losses on a nonexercise day (51). Urinary zinc excretion studied longitudinally in men indicates an acute increase in zinc output on the day of exercise with a decrease to preexercise values on the day after the exercise bout (52). Thus, acute responses in urinary zinc excretion to a single bout of exercise reflect homeostatic regulation. In contrast, urinary zinc excretion increased significantly after 1 wk and remained elevated in soldiers participating in maneuvers for >30 d (53), whereas serum zinc concentrations decreased slightly. The changes in urinary zinc output apparently reflect increased skeletal muscle turnover.

The combination of exercise and hot environmental conditions can increase surface loss of zinc. In a study by Jacob et al (54), estimates of basal surface zinc loss in men consuming ≈ 13 mg Zn/d and not participating in vigorous activity were variable at $\approx 12 \mu\text{mol/d}$ (0.8 mg/d) and represented $\approx 5\%$ of daily zinc intake. In another study, Consolozio (27) found that men participating in 30 min of submaximal work and with daily heat exposures of 7.5 h at 37.8°C (100°F) for 18 d had, with acclimation to the heat, significant decreases in zinc losses (estimated from collections of arm sweat) ranging from 209 mmol/d (13.7 mg/d) to 34 mmol/d (2.2 mg/d).

Zinc supplementation and performance

The effect of zinc supplementation on muscle function has been examined. Krotkiewski et al (55) evaluated muscle strength and endurance in 16 women supplemented with 135 mg Zn/d for 14 d in a double-blind, crossover experiment. Subjects supplemented with zinc, compared with placebo, had significantly higher dynamic isokinetic strength at $180^\circ/\text{s}$ of angular velocity and isometric endurance. The authors suggested that zinc supplementation may be beneficial in improving performance that requires recruitment of fast-twitch muscle fibers. Because the zinc status of the participants was not evaluated, it is unclear whether the supplemental zinc exerted a nutritional or a pharmacologic effect on the measured performance. Also, a 14-d treatment period does not seem adequate to replete tissue zinc stores if a zinc-deficient state was present.

CHROMIUM

In contrast with magnesium and zinc, for which metabolic functions are well defined, the biological role of chromium is not

well established except for its involvement in carbohydrate and lipid metabolism (7, 8). Recently, however, it was hypothesized that chromium, in conjunction with a low molecular weight protein, acts as a constituent of a novel insulin-signaling amplification mechanism (9). This hypothesis has not been clearly defined in humans, but signs of marginal chromium deficiency in rodents include impaired glucose tolerance, increased circulating insulin, and elevated cholesterol and triacylglycerol (56).

Dietary intake

Although chromium is unequivocally recognized as an essential nutrient, there is no RDA for chromium. The estimated safe and adequate daily dietary intake of chromium for adults is 50–200 $\mu\text{g/d}$ (13). Routine estimations of usual chromium intake from self-selected diets are limited by the lack of appropriate food composition databases and the difficulty of analyzing foods for chromium because of the low concentrations in most foods and the problem of environmental contamination.

Recent analyses of self-selected diets in the United States indicate that nutritionally balanced diets contain variable amounts of chromium ranging from 1.2–1.4 ng Cr/MJ (5–6 $\mu\text{g}/1000$ kcal) (8) to 3.6 ng Cr/MJ (57) based on the foods consumed. Other reported daily intakes of chromium in diets include 29 μg in Finland, 56 μg in Canada, 25 μg in England, and 37 and 28 μg in the United States. (8). Data on the chromium intakes of physically active persons per se are not available. Nielsen proposed that because overt signs of chromium deficiency are not present at a chromium intake of 50 $\mu\text{g/d}$, this amount may be adequate to ensure good health in most individuals (8).

Physical activity and chromium

Recent observations suggest that chromium needs may be increased by endurance exercise. Serum chromium concentrations in male runners increased immediately after a 10-km (6.2-mile) run at peak intensity and remained elevated 2 h after completion of the run (52). Also, daily urinary chromium output of runners was higher on the day of the run than on the days before and after the run. In another study, among runners who were fed a controlled diet containing 2.1 ng Cr/MJ (9 $\mu\text{g}/1000$ kcal), basal urinary chromium losses were significantly less than those observed in nonexercising men fed the same diet (58). This finding suggests that either chronic exercise is associated with a partial depletion of body chromium stores, or it results in a redistribution of chromium that is manifested by conservation of chromium by the body. In endurance-trained rats fed rat feed with a chromium content of ≈ 2 mg/kg, the chromium concentration in the heart and kidney was increased significantly, but gastrocnemius chromium concentration was not affected (59).

Chromium supplementation has been suggested to promote muscle accretion and fat loss. Studies in livestock report that chromium supplements in the form of chromium picolinate improve carcass composition. Increases in muscle area and percentage of muscle in the longissimus muscle of growing pigs were reported when the diet was supplemented with 100 or 200 μg Cr/kg diet (60). However, in another study, 200 μg Cr/kg as chromium picolinate did not affect carcass density but significantly decreased 10th rib fat and had a tendency to increase regional measures of muscularity (61).

Studies of chromium picolinate supplementation in humans have yielded equivocal results (Table 5). Evans (10) reported that young men (sedentary and football players) participating in



TABLE 5
Reported effects of chromium supplementation on body composition of adults participating in resistance training

Study and assessment method	Treatment effect		
	BM ¹	FFM	FM
	kg	kg	kg
Evans, 1989 (10)			
Anthropometry (<i>n</i> = 10 M) ²	2.2	1.6	NA
Anthropometry (<i>n</i> = 31 M) ³	−1.2	2.6	−3.4
Hasten et al, 1992 (62)			
Anthropometry (<i>n</i> = 37 M)	0.8	NSD	NSD
Anthropometry (<i>n</i> = 22 F)	2.5	NSD	NSD
Clancy et al, 1994 (63)			
Anthropometry (<i>n</i> = 36 M)	NA	NSD	NSD
Hallmark et al, 1996 (64)			
Hydrodensitometry (<i>n</i> = 16 M)	NSD	NSD	NSD
Lukaski et al, 1996 (65)			
Dual-energy X-ray absorptiometry (<i>n</i> = 36 M)	NSD	NSD	NSD
Campbell et al, 1999 (66)			
Hydrodensitometry (<i>n</i> = 18 M)	NSD	NSD	NSD

¹BM, body mass; FFM, fat-free mass; FM, fat mass; NA, not available; NSD, not significantly different from placebo-treated group.
²Nontraining men.
³Football players.

resistance training and supplemented with 200 µg Cr/d as chromium picolinate increased lean body mass and decreased fat mass compared with nonsupplemented men who were also involved in resistance training. The effect of chromium supplementation on strength gain was not reported.

Other investigators have been unable to confirm these initial results of chromium supplementation on changes in body composition. In men and women who were beginning students in weight training, Hasten et al (62) reported significant increases in the sum of 3 limb circumferences and significant decreases in the sum of 3 skinfold thicknesses as a result of physical activity. They found no effect of chromium versus placebo on strength in the men or women. Chromium supplementation, as compared with placebo, significantly increased body mass in the women with no effect on body composition. In another study of collegiate football players, Clancy et al (63) reported that chromium supplementation had no beneficial effect on body-composition change, assessed with anthropometry, or on strength gain.

More recent studies that used other methods of body-composition assessment also have not shown beneficial effects of chromium picolinate on body structure and function. Chromium supplementation, as compared with placebo, of collegiate men involved in resistance training did not promote a significant increase in strength or body composition, determined by densitometry (64). An examination of the effects of the chemical form of chromium, as picolinate and chloride, and progressive resistance training in young men also failed to identify beneficial effects of chromium, regardless of chemical form, on body composition and strength (65). Similarly, older men, aged 56–69 y, supplemented daily with 924 µg Cr as chromium picolinate, showed no significant change in body composition or strength gain when compared with placebo-treated control subjects (66). Thus, the limited studies to date indicate that chromium supplements do not promote general muscle gain and fat loss, as determined by various methods of body-composition assessment, nor do they facilitate regional or whole-body strength gain during resistance training (67).

ADVERSE EFFECTS OF MINERAL SUPPLEMENT USE

The general use of single-mineral supplements is not recommended unless an individual is under the guidance of a physician or a registered dietitian. Indiscriminate use of a mineral supplement can lead to adverse nutritional and health consequences that depend on the amount of the supplement and the duration of its use.

Consumption of magnesium supplements in amounts exceeding 500 mg/d often results in gastrointestinal disturbances, including diarrhea, and may induce a net loss of phosphate from the body (68). In addition, magnesium supplementation has been reported to impair iron and zinc nutriture in some female athletes (69).

Supplemental zinc may elicit some significant metabolic aberrations, including an inhibition of copper absorption from the diet, even if the zinc supplement is taken independently of meals (70). Zinc supplements in excess of 50 mg/d can induce copper deficiency in humans (71). Prolonged use of large doses of zinc can decrease HDL-cholesterol concentrations in men (72). In addition, use of zinc supplements ranging from 17–50 mg/d is sufficient to attenuate the exercise-induced increase in HDL concentration in men (73). Women receiving supplemental zinc in doses ranging from 15–100 mg had a significant decrease (8–9%) in HDL but only at the 100-mg/d dose (74). Thus, it has been recommended that if zinc supplements are consumed, the amount of zinc ingested should not exceed 15 mg/d (73).

Preliminary evidence for potential adverse effects of chromium supplementation is available. Chromium and iron compete for binding on transferrin (75, 76). In young men supplemented with chromium picolinate (180 µg Cr/d for 8 wk), there was a 28% decrease in transferrin saturation (65); a similar trend was not evident among the men supplemented with the same amount of chromium as chromium chloride. Additional research is needed to clarify the magnitude and impact of this interaction.

Scientists who study chromium have expressed concern regarding the potential effects of chromium picolinate supplementation on tissue chromium accumulation and toxic side effects (77, 78). The use of chromium supplements in excessive doses or for prolonged durations is not advised because the long-term biological effects of chromium accumulation in humans are not well understood (67).

SUMMARY AND CONCLUSION

The allure of supplemental magnesium, zinc, and chromium as a way to improve well-being and enhance physical performance should be viewed with caution. Experimental evidence indicates that acute physical activity increases loss of minerals but that these losses are transient. Furthermore, when dietary intake of the mineral elements is consistent with population guidelines, there is no alteration in mineral status as evidenced by plasma or serum concentrations.

Evidence of beneficial effects of generalized supplementation of minerals is equivocal. Whereas some studies indicate improvement in physical performance with supplementation, the nutritional status of the participants is inadequately documented. The failure to assess nutritional status of subjects, particularly for the mineral under study, precludes a determination of whether the effect of supplementation is attributable to a nutritional deficit or to a pharmacologic response.


Evaluation of the interaction between diet and physical activity and its effect on nutritional status is needed. To meet this goal, more sophisticated experimental designs are required. Studies

should require assessment of dietary intake, blood biochemical studies of circulating minerals and mineral-dependent enzymatic activities, and sensitive indicators of body mineral pools. With this baseline information, researchers can evaluate the influence of diet and supplements on physical performance. However, previous trials of magnesium, zinc, and chromium supplementation of physically active individuals with apparently adequate nutritional status have not shown any beneficial effect of supplementation on either nutritional status or performance (79, 80). Thus, supplementation with these minerals has no apparent ergogenic effect in individuals consuming diets adequate in essential nutrients.

RESEARCH NEEDED

A variety of experimental approaches may be used to delineate the biological roles of magnesium, zinc, and chromium in physical activity. The most stringent experimental approach uses controlled feeding studies in which the mineral element of choice is provided in graded amounts from different food sources. Volunteers consume a basal diet that is low in the mineral under consideration and then receive the same diet that is supplemented with the mineral in question. This depletion-repletion design includes a washout period between the experimental treatments and is amenable to a randomization of treatments to avoid time effects. Alternatively, one might survey physically active persons and identify those individuals with blood biochemical measurements that are indicative of mineral depletion. The mineral-depleted individuals would receive either a mineral supplement or a placebo for a defined period of time with the possibility of a crossover in treatments.

Any experimental design with mineral intake as a treatment will benefit from additional measurements of mineral metabolism and biological function. Because mineral intake is controlled, measurements of mineral losses in urine are required to estimate turnover. In addition, measurements of mineral-dependent enzyme activities would be informative as indexes of cellular mineral nutriture. There also is a requirement to assess physiologic functions at rest and during controlled stressors such as submaximal work and actual performance. This approach permits an evaluation of the influence of mineral status on ability to perform under conditions that a physically active individual would encounter. Another aspect of the interaction between mineral nutriture and physical activity, and one area that has not received adequate attention, is the effect of intake on recovery during chronic physical activity or training.

A key limitation in delineating mineral nutrient requirements for optimal biological function is the lack of an integrated and multidimensional experimental design. Using this approach requires control of nutrient intake, assessment of daily losses, and measurement of physiologic and psychological functions under different amounts of stressors. A fundamental consideration in the implementation of this approach is the use of physically trained individuals and multiple stressors to gain insight into the metabolic and psychological adaptations that might be influenced by graded mineral intakes. Participation by less well-trained individuals also has the potential to highlight new biological roles of minerals. However, factors such as familiarity with the demands of routine exercise, particularly activities with high-intensity demands, may restrict the ability to identify physical and psychological impairments and advances because of the variability in the responses in the less-adapted individuals. 

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